

MULTIPLE FALLACIES IN THE CONCEPT OF SCHIZOPHRENIA

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I. PREFACE

At last a rebellion against the concept of schizophrenia is in full swing. Recently many psychiatrists are gradually coming to realize that this combination of a misconception and a misnomer has misled us up blind alleys. Many who seem to be fearful about writing about this are talking about it timidly but freely. In a conference at the Menninger Foundation in the winter of 1968-1969, some of the discussants openly expressed their doubts as to the validity of the concept. For example, Altschule said flatly, "In fact the word schizophrenia should be abandoned". Then he proceeded to list several compelling reasons for this. It is well known that for some years Karl Menninger has taken a comparable position. Cancro and his co-workers wrote with humor and scorn of the "mighty role of the Latin phrase 'dementia praecox'", and again of the "Greek term 'schizophrenia'", as "manufactured in Switzerland and later duly latinized for English speaking consumers". Then they ask whether if "neither term had ever been coined and used, would the linguistic loss have thwarted our ability to see madness when it occurs, or would it have curbed our attempts at understanding it?" Other authors were less ready to abandon the concept: yet it is clear that there is a slow but general awakening to its multiple fallacies. Our emancipation from it will free psychiatry for important clinical and conceptual

advances and for greater clarity in research. Yet the concept of schizophrenia is only the most outstanding example of the deficiencies of the psychiatrist as a naturalist in his own field. What I will write here applies in varying degree to all psychiatric nosology.

To preclude misunderstanding, I will state at once that my thesis has nothing in common with the position for which Dr. Thomas Szasz (15) is well known. He doubts that there is any such thing as a psychological "illness" and calls mental illness a "myth". Contrariwise, I affirm the existence of mental illnesses as processes, in spite of the fact that we do not yet know how to differentiate simply and clearly among them or to separate them into discrete clinical entities. Whereas Szasz denies their existence, I deplore our bondage to misleading pseudo-diagnoses inherited from the past. In fact, it is my impression that Szasz has made the mistake of misunderstanding his own position; and that what he is really denying is the accuracy of efforts to characterize, identify, isolate and diagnose discrete processes of mental diseases. This position is sound; and he would have made a useful contribution if he had limited himself to objecting to the pseudo-nosology which has resulted from the failure of psychiatrists to be better naturalists, and if he had also pointed out that in psychiatry much of that to which we have given names, which imply that they are separate "illnesses", are in fact only transient cross sections of long and constantly changing processes, cross sections which are marked by clusters of symptoms. It is essential to add that clusters of symptoms are important, but not because they result from uniform processes of illness with common origins and similar courses, but rather because the destructive consequences of specific

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symptoms and symptom clusters are predictably and relatively constant. This is true for somatic as well as psychological medicine. For example, alcoholism can arise in many types of psychological soil, with no single somatic or psychological origin or cause or precipitating trigger. Yet its destructive consequences for a human life and personality are *relatively* so constant that ultimately most alcoholics of similar degrees come to look more alike than different. There are also important differences which are due to differing secondary consequences. Some alcoholics consistently develop fighting jags; others cry; others become euphoric; other alcoholics develop terror states. Some are strikingly stereotyped; others vary from one occasion to another. (In this respect, they call to mind the varied affective colorings of different dreams in the same individual.) Such variations have a profoundly different impact on a life and of course on all human relationships; but in the end of the recurrence of similar secondary effects in the lives of alcoholics is responsible for the tendency to look upon alcoholism as a disease *sui generis*. The same can be said of all clusters of neurotic symptoms. However different its origins may be, any specific neurotic symptom has relatively constant and predictable consequences, which ultimately impose on life an apparent uniformity. Of course, some come to look more alike than others; but to regard them as units of illness simply because the destructive consequences of their symptoms are relatively similar is a fallacy as obvious as it is prevalent.

On the other hand, our failure to differentiate accurately among the varied sequences and processes by which men become and remain psychologically ill, or to characterize these sequences with precision does not mean that there are no units of illness in psychopathology. The history of somatic medicine is full of similar confu-

sions; but this has never led anyone to deny the existence of discrete somatic illnesses.²

II. THE FALLACY OF DIAGNOSIS BY SYMPTOM CLUSTERS

In his lectures to medical students, Adolf Meyer would often point to the fact that a nosological system which is based on symptom clusters leads to inappropriate diagnostic "fashions", which have always exercised a confusing influence in psychiatry. He would present statistics to show that during the last decades of the 19th century there were years in which a large majority of the patients in some German hospitals would be called "dementia praecox", while during other years in the same hospitals approximately similar percentages of patients would be called "manic depressive". These discrepancies made him doubt that either constituted a disease entity. Yet, his emancipation from symptom clusters was never complete. In fact, many years later he formulated a nosological system of his own which had its roots in symptom clusters. He coined a new term and called it "*Ergasias*", in a fallacious analogy to the concepts of units of energy (*i.e.*, "ergs") which were then current in physics. Despite these limitations, his neologism had the beneficial effect of partially freeing Meyer himself (and to a lesser extent American psychia-

² Before going into details I would point out that the most misleading component in the word which characterizes the concept of schizophrenia is the syllable *schiz*, implying as it does a schism. This syllable has been seized upon in popular writing about this condition and in technical writing with the implication that this schism is unique for this condition, whereas, as a matter of fact, there is no form of psychopathology which does not involve a schizmatic process. This was pointed out in a paper by Margolin and myself published in 1945 (The therapeutic role of drugs in the process of repression, dissociation and synthesis [with Margolin, S. G.]. *Psychosom. Med.*, 7: 147-151, 1945.). In this article the fact is emphasized that dissociative processes are inherent in the dynamics of every form of psychopathology. Therefore, to single out a schizmatic process in the term "schizophrenia" leads us back to an erroneous assumption.

try) from the rigidities of the Kraepelinian system which still dominated European psychiatry at the time.

Bleuler had also reacted against the symptom clusters which were incorporated in the Kraepelinian dogma; but like Meyer he ended by merely substituting a different terminology also based on symptoms. Out of this he derived the concept of "schizophrenia", which has come to exercise an equally restrictive influence on psychiatric observations, thinking and progress. Although its implications are somewhat freer than Kraepelin's, it embodied most of its predecessor's fallacies, and remains as ill-defined clinically as when it was first formulated. Unfortunately, this has not prevented its becoming the fashionable "diagnosis". The tendency now is to forget that there are such things as chronic neuroses, which produce distortions in personality and in patterns of living, block human relationships and resist treatment. Instead, almost any tenacious neurotic illness is labelled "schizophrenic" or "schizoid" or "pseudo-neurotic schizophrenia" or "borderline". In some measure, the responsibility for this confusion rests on Bleuler himself; because his original clinical descriptions of the patients he called "schizophrenic" included many who should not have been considered psychotic in any sense. This made it an ambiguous concept which side-stepped all critical issues. Therefore, it is especially unfortunate that psychiatrists, young and old, have actually become afraid *not* to use the term, lest some colleague point a scornful finger and say, "He does not even recognize schizophrenia". This has resulted in a timid conformity which has blunted our clinical perceptions and paralyzed our clinical thinking by freezing it into rigid molds, rendering our clinical language so stereotyped as to be almost meaningless. Assumptions which are dominated by over-riding preconceptions lead to pseudo-perceptions. These in turn lead to the recording of stereotyped clinical

histories and examinations and to descriptions which are shaped to fit our preconceptions. Therefore, if we continue to accept the concept of schizophrenia uncritically, our clinical discriminations will remain dulled and our clinical histories and reports of mental status examinations will be cliché-ridden. It is especially unfortunate that the clinical perceptions and formulations of our senior psychiatrists and clinical psychologists have been blunted by this stereotype; because the effects of this have trickled down to impede the growth of our residents.

If any patient is slow to respond to therapy, and is hostile, shy and awkward or off-beat, to call him "schizophrenic" may help us to feel more comfortable about our therapeutic failures. The formula is "I failed because the patient was really schizophrenic all along", or a "pseudo-neurotic schizophrenia". Such a formulation blocks progress and leads to a parody of psychodiagnoses. Unless every isolated psychotic episode is a disease process, it is a preposterous fallacy to place in this category every patient who has ever had an episode of psychotic disorganization.

III. ILLNESS AS A PROCESS³

All illnesses are processes. Whether they are somatic or psychological or both, chains of cybernetic relationships develop between the changes in different components of the individual's somatic processes, their interactions with one another and also with the

³ A great deal has been written recently about the supposed differences between a form of illness known as reactive schizophrenia and the form known as process schizophrenia. One could draw comparisons between this and the concept of reactive depression and a depressive process; but if one acknowledges the fact that all illnesses are processes and that all processes have reactive components the significance of this distinction becomes dubious. If, in addition, one doubts that there is a form of psychosis which must be differentiated from the others by a distinguishing name, *e.g.*, schizophrenia, then there would seem to be little reason for the distinction at all. For these reasons I will not enter into this controversy.

outside world. For example, the total process of illness in a patient with incapacitating heart disease involves not only his heart and his circulation and his renal functions and the movement of blood through his lungs and other tissues and the supply to the tissues of oxygen, fluid, salts, and food-stuffs and the removal of carbon dioxide and fluid, salt and unused food residues, but also his role in his family, his human relationships in general, the economic security of himself and his family, the effects of all of this on changes in his affective and cognitive psychological states and processes, etc. But this also involves, if less directly, changes in the attitudes to him of family, friends, employers and employees and in turn his responses to these changes. Variations in vulnerability must also be included, possible genetic and psychological determinants, and of course the after-effects of any persisting structural and/or psychological changes and deficits. Clearly, every sickness is a combination of variations in biological, psychological and psychosocial processes, the consequent psychosocial and socio-economic distortions, and also the interactions among these changing processes which combine to produce a sick life. There is an important distinction between the initial process which is the core of the illness and the sick life which gradually results and which may persist long after the sickness as an active process of pathology is over. The sick life of a quadriplegic will last for many years after the active infection with the virus of poliomyelitis has disappeared.

To at least an equal degree, or perhaps even more so, these phases are recognizable in the processes of psychological illness. Here a chain of cybernetic relationships evolves between changes in the "I" world and the "non-I" world, in their interactions with one another and with psychological objects, figures, relationships and events in the inner and outer worlds.

For instance, the ultimate consequences

of a height phobia may be complicated and devastating to a man's conceptual, perceptual, cognitive and affective processes, to his relationships to family and friends and work associates, to his work itself, and gradually to his whole life and personality. In his efforts to escape the fear which is evoked by any situation which might compel him to confront a height, he may develop hypochondriacal and also hysterical distortions, illusions of body change, humiliation, depression and fabrications, fears of their detection which lead to suspicious attitudes, thus giving a paranoid coloring to his illness. None of these elements can be omitted from our conceptualizations of psychological illness as a process of progressive distortions, which are first produced by the symptoms themselves and then by the patient's battle against his symptoms. Furthermore, these secondary distortions produce new conflicts, new symptoms and new levels of distortion.

Several years ago, I pointed out that several ingredients are essential for the characterization of any process of neurotic illness (3, 6, 7). Some years later, I traced their relationship to psychotic disorganization. I will review these here, but add to the original list (11).

1) There must be a neurotic potential which is truly universal, but which has several variable components and determinants, which may vary from individual to individual. What constitutes the neurotic potential will be itemized below.

2) Out of the neurotic potential and under the influence of differing biophysical, biochemical, genetic, familial, experiential, developmental, intrapsychic and socio-economic forces, a variable progression of events will follow in different sequences. To the extent to which this variable progression is determined predominantly by psychological processes of which the individual is not only unaware but also unable to become conscious without special help, this march of events constitutes the neurotic

process: because wherever unconscious determinants play the dominant role, they will predetermine the automatic repetition of that behavioral pattern, even if the external conditions have changed.

3) Furthermore, from time to time and under appropriate circumstances, a neurotic state may precipitate out of the evolving neurotic process. This will be manifested in varied clusters of old and new overt symptoms. These characterize the neurotic states but never in themselves constitute the disease process. Symptom clusters are only cross-sections in the long-drawn out process which constitutes the illness. It may at times have some practical value to give descriptive names to these symptom clusters, e.g., phobia, compulsion, conversion, etc., etc.; but the symptom cluster remains only a highly charged cross-section in an illness, but not the process of illness.

4) The symptom clusters have another and more important significance which psychiatrists generally, myself included, have tended to overlook: namely, that the symptoms themselves produce distortions. These secondary consequences cause new distortions of lives and of personalities in many ways. For instance, the far-reaching secondary consequences of a claustrophobia are different from those of an agoraphobia, although the two are closely related. The processes of progressive disorganization which each can initiate are different. The same is true of a *Sozial-Angst* versus a terror of being alone; or of an eating compulsion and an anorexia nervosa. What is more, out of these secondary distortions new conflicts and new symptoms arise, which in turn produce new tertiary distortions and a third order of symptoms.

5) Subsequently, I (11) pointed out that under other appropriate circumstances and stresses, these sequences may lead to an insoluble impasse, out of which psychotic disorganization may supervene. For a variety of secondary reasons, this sometimes occurs with dramatic suddenness, at other times as

the result of a slow accumulation of subtle increments. This is another clinical bifurcation whose consequences can produce remarkably divergent clinical pictures, even within a single process of illness.

In all of this, the basic fact is that whenever new symptoms appear at any step in the sequences of an illness, they distort life in new ways, producing new conflicts, new symptoms and new distortions. This is how long chains of neurotic distortions may form, linked in an unending sequence of cybernetic circuits, new conflicts, new symptoms, consequent distortions and then new conflicts, symptoms and distortions with the constant threat of the precipitation of new symptomatic nodal points all along the way. These reverberating chains constitute the process by which psychological illnesses become self-sustaining.

IV. INGREDIENTS IN THE NEUROTIC PROCESS AND PSYCHOTIC DISORGANIZATION

In 1963 Dr. Eugene Brody (1), in an article entitled *From Schizophrenic to Homosexual* described a patient whom he had treated. This is valuable and challenging and relevant to my thesis. The patient's illness passed through many phases, to each of which a different label could have been attached, because in each the intensity and quality of the manifest symptoms varied. Some phases were dominated by fear, guilt-laden homosexual fantasies, masochistic fantasies and practices, anal practices, and some unclear paranoid feelings about his mother. His high academic achievement was unimpaired through professional school, but with increasing isolation and one or two incidents of unclear auditory hallucinations and fear of becoming psychotic. All of this finally led to hospitalization, with improvement to a point at which he could return to intensive psychoanalytic outpatient therapy. The psychotic symptoms disappeared and most of the neurotic anxiety and inhibitions as well, as he re-es-

tablished his professional life and worked through potency problems in a homosexual adjustment. The remaining deficit can best be described as "characterological" rather than either neurotic or psychotic.

A study of a comparable sequence in a child of about five was first published in *The Psychoanalytic Study of the Child* under the title "Say you're sorry" (14). Here the transit from neurosis to psychotic disorganization and back into the neurotic illness on the way to health is clearly depicted. Subsequent developments are described in a later publication (10).

In the paper entitled "The relationship of the neurotic process to psychotic disorganization", several comparable case vignettes are described (11). In not one of these would the use of the term "schizophrenia" have been descriptively accurate, illuminating, necessary or even pragmatically useful.

In 1951, I published an article entitled, "The neurotic potential, the neurotic process, and the neurotic state" (3), in which I emphasized the importance of recognizing these as linked steps in the development of a neurotic illness. I have pointed out that while it is possible and justifiable to characterize an individual moment of behavior as normal or neurotic (or psychotic), the application of the same terms to a whole individual or personality or to an entire life is never permissible. Doing so merely turns the words into epithets, instead of diagnoses or clear characterizations of a process or phase of illness. Actually if the word "normal" is similarly misused to characterize a whole man or a whole life, it becomes merely an affective accolade, the expression of a personal preference. Yet, we can apply these same words with precision to individual moments or episodes of behavior. For example, we can say of any moment of thought, planning, anticipation, reflection, feeling or overt behavior, that if the constellation of processes which determined that moment predetermined its automatic repetition, irrespective of pain or pleasure,

reward or punishment, success or failure, satiation or need, that kind of behavior would differ from "normal" behavior, in these significant aspects (2).

Psychopathological behavior is always the product of mechanisms that deprive the subject of the freedom to learn from experience and to change under the impact of changes in the cues that reach him, either from within or from without or both. It cannot cease even when sated. It is this loss of the freedom to change which is the essential core of the psychopathological process and it is this loss of freedom which has predictable, characteristic and distorting consequences. It has also been pointed out that somatic as well as psychological variables can exert an influence here (7).

When, on the other hand, the constellation of multiple determinants of any pattern of behavior leaves that pattern free to change in response to changes in the cues which reach the individual from changes in his internal and/or external milieu, we are justified in calling this pattern of behavior "normal" (5, 6).

In short, the heart of the matter is in the effects of the determinants of behavior on the freedom to change in response to changing input. Any constellation of *psychological* processes which predetermines the automatic, stereotyped and obligatory repetition of behavior and which reduces or limits its freedom to change is pathogenic: *i.e.*, neurotogenic, or psychotogenic, whether the variables are somatic or psychological or both.

Today, I would expand my view of the nature of neurotogenic determinants to include some which I failed to include in 1951. My conception of the ingredients which make up the neurotic potential and process and state has grown over the years. Originally, I emphasized some degree of the capacity for perceptual accuracy, of the ability to generalize from multiple experiences (which implies some accuracy in the recognition of similarities and dissimilarity-

ties), the ability to abstract and generalize from comparable units of experience and to represent these abstractions by symbols. Today I would add the ability to process and digest such experiences preconssciously and then to sample the continuous preconscious stream and to represent these samples by consistent symbols (4).

These are essential ingredients in the thinking processes of those who are not suffering from severe degrees of mental defect.

Today, I would also give more attention both to the early molding of the personality and of behavior into what I have called a "central affective potential" (8) and also the later evolution of this into a "central affective position", plus the secondary consequences of these. Also today, I would place greater emphasis on the ways in which even the early steps in the neurotic process can block all maturational processes. This again has many further secondary consequences (12). Finally, I would stress today the hampering and distorting influence of neurotogenic symptoms themselves on the development of a patient's personality; *e.g.*, how they generate obstacles to his free use of his native abilities, thus exposing him to many defeats with resulting affective upsets, which expose him furthermore to the impact of new conflicts, affects and symptoms which such defeats inevitably produce. In short, I would emphasize today the enormous importance of the secondary and tertiary distortions which result from even simple neurotic symptoms. This view of the psychopathological process sees it as a continuous feedback of destructive consequences, which evolve out of the original neurotic disturbance in a cybernetic chain of secondary and tertiary distortions.

I am afraid that as psychiatrists we have tended to overlook the fact that neurotic symptoms themselves, whether overt or masked, have destructive consequences; and that in general the masked symptoms which are so rarely brought to light in the

standard anamnesis have at least as destructive secondary consequences as do those symptoms which are overt. In fact, the initial symptoms which start the ball rolling down hill may themselves be so minor as to be overlooked (*e.g.*, an exaggeration of ordinary shyness, or a masked depression or phobia on going to kindergarten.) Yet, such an anxious depressive shyness may persist throughout school, college and even doctoral and post-doctoral education until the whole life may become far "sicker" than the original "sickness" itself: just as the life of a quadriplegic remains a sick life long after the disappearance of the infective poliomyelitis which originally damaged the spinal cord. This leads to one other basic distinction; *i.e.*, that between the severity of a neurosis as a process of illness and the degree of "sickness" of the life which it produces. Our official nosology consistently ignores the latter and confuses it with the former.

The main point of all of this is that the phenomenology which has been subsumed under the general concept of "neurotic" must be broken down into a succession of interdependent and interrelated yet separate steps. Until and unless this is done, our efforts to establish correlations with other variables (whether somatic, experiential or psycho-social) will fail; because the only correlations that can be meaningful will be between the individual variables, the thresholds of vulnerability, and the transitions from one to another in the evolution of the neurotic process; never merely with the end-result; *i.e.*, with that ill-defined, overall abstraction which is what is usually and erroneously called "a neurosis".

Certain additional distortions are needed to warrant the use of the term "psychotic". Among these are disturbances in the ability to make generalizations from multiple experiences, disturbances in the ability to represent these generalizations as abstractions by the use of symbolic tools and devices, the resulting distortion in the ability to

build conceptual abstractions, the distortions in the disruption of the relation of all symbols to their referents, and the regressive consequences of all of this.

Recently, I have described one of the ways in which an impasse in the neurotic process can lead directly to psychotic disorganization (10). Any attempts to establish correlations between the *final* stage of psychotic disorganization and biological or psychosocial variables would only be misleading. These must be sought in the steps by which the psychotic process as such originated and then evolved the thresholds of the successive steps of change, but not with the end state.

Various degrees of psychotic disorganization can be recognized as the outcome of a psychotic potential and process, leading to the psychotic state. Correlations can be sought between somatic and/or experiential variables and the steps in this process. But within this general concept of progressive psychotic disorganization, there is no need (or justification) for a separate sub-category among psychotic disturbances to be called "schizophrenia" or "schizoid". I am more convinced of this today than ever, and I challenge my colleagues to prove its necessity or its usefulness, and to demonstrate that it does not add to the confusions of our clinical descriptions and of our research.

V. FURTHER CONFUSIONS INTRODUCED BY THE PRESENT STATE OF PSYCHIATRY

Often, we inadvertently introduce distortions of behavior into psychopathological syndromes, thus increasing the difficulty of separating out those components which are exogenous from those which are endogenous to the patient and also intrinsic to the essential process of illness. This compels us to re-examine the influence which is exerted on the process of illness by the patient's family, his job, his living situation, his friends, his medical and psychiatric condition as

well as our cultural institutions and "therapeutic" facilities.

Old photographs of patients in psychiatric hospitals raise unhappy memories and images and questions: *e.g.*, disheveled patients, half-clothed in hospital shifts, either rejecting all efforts to care for them or sometimes neglected for long inert hours; seated on hard benches along a wall, knees drawn up, staring at the floor in attitudes of total dejection or abstraction; seemingly mute and absorbed in nothing. The better organization of hospital care today attempts to eliminate this; but these ancient impressions still color our thinking about the nature of chronic psychological illness and its origins, by whatever name it is known. They still influence our feelings about today's psychotic, our expectations as to what we will see and hear, and therefore how we distort and digest our perceptions; how we classify our impressions and the language in which we express them. We have to ask ourselves whether and to what extent the behavior of these patients constitutes an unwitting defensive effort to obliterate the misery of their external circumstances; or to what extent they are defenses against internally generated conflicts, impulses and painful feelings. Consequently, as we reconsider the validity of the concept of schizophrenia, we have to consider whether and to what extent we ourselves contaminate the very clinical data on which our efforts at clinical categorization are based.

For example, when I am told that these patients sometimes soil themselves and sometimes require tube-feeding or that they were "hearing voices" and "seeing things" (with or without conclusive evidence of either), I would know how these patients had been categorized, even without hearing the histories of their lives or of the evolution of their symptoms or the form and content of the mental status examinations. Such epithets as "paranoid schizophrenic", "chronic hallucinatory state", and

"chronic undifferentiated schizophrenia" would be generally taken for granted.

Or when such a patient occasionally erupts into open and blind rebellion I know that he will be called a "catatonic excitement". Or if he consistently covers up his inner turmoil and pain with a "silly" smile, a giggle, or irrelevant childlike words or gestures, he will surely be called "hebephrenic". Of course, these terms have some pragmatic value as a descriptive verbal shorthand; but they are not diagnoses; and when such a patient emerges from this state as they do from time to time (if only temporarily), he may surprise us by talking freely and clearly about himself and his seemingly bizarre external behavior and inner experiences. When this happens, we will find ourselves wondering why anyone should ever have expected him to be anything but mute or motionless. From the patient's point of view, was there anything worth his saying or doing? Anything that he could have achieved by saying or doing? Similar double-ended questions would then have to be asked about the other items in his disturbed behavior. The question "why" he soiled should have been paired with the question "why not?" Why *should* he eat must be paired with why does he *not* eat? And since both alternatives must be weighed, if our answers are to be meaningful, not one of these questions should be asked one-sidedly. Yet, it is rare that we ask ourselves why a patient should *not* have some symptoms.

These questions do not imply that every such symptom is always under voluntary control, and that the patient is always free to turn them on and off at will; but only that more of the picture is at least partially volitional than is usually realized, and that much of it is determined by our influence, and by combinations of preconscious and conscious purposes, altered perhaps by unconscious symbolic processes but not dominated solely by unconscious mechanisms.

Yet, because these alternative possibili-

ties are not usually realized, we affix to such patterns of behavior names which imply that the behavior is immutable and that the dominant roots and continuing determinants are to be found in genetic or biochemical anomalies, in some idiosyncratic pattern of buried experiences and in the unconscious developmental conflicts which result. When we make these assumptions, we shut our eyes to the significance of many well-known clinical experiences; such as the fact that when that same patient breaks a leg or suffers pain of any kind, the cluster of his disturbed symptoms and disturbed behavior will often melt away, only to return again as the body recovers and the pain recedes. This familiar sequence of events should lead to the obvious conclusion that, whatever the nature of the variables which determine both the vulnerability to psychotic disorganization and also its tenacity, they are not immutable but are modifiable by other psychological and somatic variables. Yet, so "alien" is the patient's behavior when he seems most disturbed that it took us many generations to learn that the hospital environment, the behavior towards the patient of doctors, ward personnel and other patients play important contributory roles in shaping the pattern of the patient's symptoms. Furthermore, it still is not realized that the after-images of the patient's own behavior during some earlier disturbed episode, however brief, can provide a patient with material for subsequent "hysterical" facsimiles of psychotic disorganization. Along this neurotic path, patterns of pseudo-psychotic behavior can emerge out of the neurotic process as manifestations of a conversion process which is making use of pseudo-psychotic patterns (9). In short, we have shut our eyes to the multiple and powerful iatrogenic influences of our behavior, of the earlier behavior of the patient himself, of the behavior of other patients, and in general of the environments in which patients live out their sick lives. These confuse the clinical picture with extraneous

contaminants, which not only make precise diagnosis difficult, but also make it difficult to decide what and when and where to seek correlations.

At the same time, we have also misled ourselves by pretending (to ourselves and to others) that behavior has been more carefully observed, recorded and differentiated than was possible until the recent introduction of audio-visual aids.

The advent of modern devices for audio-visual recording and play-back makes a new era possible, but only if these devices are used consistently and critically. If we are ever to solve those problems, audio-visual aids must be used on every patient whose record is to be accepted as dependable scientific data. Furthermore, they are needed to provide us with statistically adequate samples of permanent recordings of basic observations, which can then be subjected to repeated study by many observers. Until we do this, we will continue to depend upon fallible reports of fallible and weighted memories of our skewed perceptions of rapidly changing and emotionally charged moments of behavior in which many things were occurring simultaneously (13). This is what we have had to work with in the past. It has hardly been a foundation upon which to build an accurate nosological system.

For centuries, this limitation blunted our discriminations, our perceptions and our sensibilities and at the same time blocked our compassion. It enabled us to avoid asking ourselves such uncomfortable questions as what we were doing to a patient to confirm and justify to him his secret feelings of guilt, his buried resentments, his vague feelings of persecution. Only recently are we asking ourselves how much of his illness have we reinforced and entrenched if not created, thus alienating the patient still further and deepening in him the conviction that it is not worth his while to talk, to think, to communicate or to act, except on those occasions when he explodes with grief

or fear or rage or withdraws to regressive infantile levels of soiling and to neologistic or infantile speech, etc. As a consequence, especially with respect to all regressive components in a clinical picture, it is only recently that it has become possible to estimate whether we ourselves produce them or whether they are endogenous to the patient and an inherent part of the process of illness. Of course, many concurrent unconscious conflicts contribute to grief and fear and rage, which in turn lead to regressive trends, and thus produce distortions in a patient's affective, conceptualizing and symbolizing processes and in his relationships to others. But this only makes it all the more essential to ask ourselves to what extent these have their roots in him; or to what extent they have been generated by and have received reinforcement from the realities of the hospital environment to which we have consigned the patient. Nor can we answer such questions until and unless we compare the clinical picture as studied in a radically different environment and by basically new methods of recording and sampling.

It is on the basis of such defective "histories" and on the basis of fallible reports of fallible observations of current behavior that in the past our so-called diagnoses have had to be based. Until recently, there was no other way. Today however, if we ourselves are to become reasonably scientific clinical naturalists in psychiatry, and if we are to train others in the same model, we will have to use audio-visual reproductions of primary data, not as a substitute for total recall (like the idiot *savant*) but as sampling devices. This scientific stance will provide us for the first time with fully dependable data out of which to reconstruct the entire nosology of psychopathology.

VI. IMPLICATIONS FOR CORRELATION STUDIES

The search for "hard", "objective", quantitative data on the hypothetical concept of

schizophrenia has flooded the literature with reports on studies of correlations between something called "schizophrenia" and variations among biophysical, biochemical, physiological, genetic and endocrinologic factors, and also with variations in familial types, patterns and stresses, personality types and temperaments, and with idiosyncratic life experiences as these occurred at different ages. Until quite recently, few among us have stopped to ask ourselves whether the inconstant symptomatic clusters with which these correlations are sought constitute in fact a discrete process of illness. This basic question has been bypassed, as though an affirmative answer were self-evident. Yet it is of little value to be precise in chemistry if we then try to establish correlations between exact chemical data and a non-existent clinical abstraction. This can lead only to a continuing waste of large sums of money, laboratory space, personnel, talent, and experimental equipment. It is natural that even the ablest colleagues from other disciplines, who naturally are naive about clinical psychiatry, should accept our "diagnoses" as valid, assuming that the "schizophrenia" which is talked about with such confidence really exists. The epithet "schizophrenia" has become so entrenched that it pretends to our colleagues from other scientific disciplines that this misconception is a true clinical entity for which it is fair to ask them to seek correlations with it. Consequently, if the clinician clings to the assumption that the entity exists and never re-examines or challenges it, he will continue to mislead his fellow scientists from other disciplines, until ultimately he will lose their cooperation. In the end, this will betray not only our own hopes as psychiatrists, but also those of the representatives of other disciplines.

It was for this reason that many years ago, I repeatedly suggested to scientific friends in neurobiochemistry, genetics, soci-

ological and familial anthropology, etc., that they should make a basic change in their search for correlations. I urged them first to gather a statistically adequate and random sample of the population as a whole, gathering this sample without using any psychological variables as a basis for selection; then determining on this sample what they would consider to be adequate and dependable profiles, whether pharmacological, biochemical, genetic, cultural, etc.; and that they should then determine whether or not there is a tendency for any of these profiles to appear in clusters. If such clustering occurred, the next step would be to select, again at random, a statistically adequate sample of individuals from each cluster; and to subject these individuals to psychological studies carried out "blindly", *i.e.*, without any foreknowledge of the group from which any individual had been drawn. Experienced psychiatrists, clinical psychologists and psychiatric social workers could then gather the anamneses and the mental status examinations. Psychoanalysts could record and study their free associations under relatively constant conditions, both with and without interpretations. Experimental and clinical psychologists could apply testing devices. On the basis of these data, and without any foreknowledge of the history or nature of the patient's life or illness, any correlations which were found with somatic, sociological, familial, educational and general psychological groupings could then have meaning. Such studies would provide us with undistorted and unskewed data on which to base correlations in whose objectivity we could have confidence. Without such a re-examination and re-testing of basic methods for gathering clinical data, we have no longer any right to use an existing nosology which does not even provide adequate discrimination between relatively normal, relatively neurotic and relatively psychotic processes of illness.

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